ENDOCARDITIS WITH STREPTOCOCCEMIA.*

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Under this title it is my purpose to consider only that group of cases of the so-called class of malignant or infective endocarditis in which the streptococcus is demonstrable in the blood stream and with the particular purpose in view of translating the older conceptions of the etiology of this disease into terms of the recent explanation of its bacteriology and pathology as offered by the brilliant work of E. C. Rosenow of Chicago.

Thus, while it has long been known that malignant, infective endocarditis occurs secondarily to acute rheumatism, pneumonia and septic processes of all kinds, the relationship between the bacteriology of these various conditions as shown by Rosenow offers the underlying reasons for this intimate association. The various biologic phenomena of such individual organisms as streptococcus hemolyticus, and diplococcus pneumonia, the specificity of their immune bodies in particular, and their commonly recognized bacteriologic properties, their cultural differences, etc., have thoroughly distinguished and dissociated them one from the other in the minds of the medical profession. As a result it was easy enough to understand why secondarily to a case of pneumococcus pneumonia or in a primary case of pneumococcus septicemia we might find an infective endocarditis and recover the pneumococcus from the blood stream. It was not easily comprehensible, however, in the event of finding a streptococcus in the blood stream associated with an infective endocarditis secondary to pneumococcus lung infection or acute rheumatism, and we commonly supposed that a superimposed infection was the cause.

By the work of Rosenow we are enabled to understand clearly the reason for this apparent double infection. His work and findings may perhaps be best presented by more or less condensed abstracts from his articles. He finds that "it has been possible to transform typical encapsulated pneumococci into hemolytic streptococci and vice versa. The transformation has been so complete that I have no hesitation in saying that if bacteriologists can differentiate a typical pneumococcus from a typical hemolytic streptococcus, then it is possible to transform one into the other. The pneumococci formed from streptococci have capsules, are indistinguishable from the former in morphology, correspond to all the differential tests known, have a pathogenicity more or less characteristic of pneumococci, producing hemorrhages in lungs and exudative pneumonia of a certain range in virulence, and pneumococcemia when still more virulent; are agglutinated specifically by anti-pneumococcus sera, and give rise to the formation of antibodies specific for pneumococci. Hemolytic streptococci formed from pneumococci behave exactly as do streptococci from the usual sources. During the transition of pneumococci into streptococci, and vice versa, there are intermediate forms which correspond to those strains, of which streptococcus viridans is the most typical example, and to the streptococci from rheumatism. The apparent correct position of various streptococci may best be illustrated by a partially flexed hand in which hemolytic streptococcus occupies the position of the little finger, the pneumococcus the place of the index finger (the opposite extreme), streptococcus viridans (representing the group of more or less saprophytic, nonhemolyzing streptococci) the middle finger, streptococcus 'rheumaticus' the fourth finger, and streptococcus mucosus, partaking some of the properties of both pneumococci and streptococci, the position of the thumb. In this grouping there is in general an increase in parasitism and virulence as we approach the thumb (streptococcus mucosus). The fact that they are members of the same family is illustrated by their being members of the same hand. The sign of reversible chemical reaction $(\leftarrow\!\!-\!\!-)$ $(-\!\!-\!\!\rightarrow)$ between each member might be used to illustrate their transmutability."

It was further shown by growing hemolytic streptococcus in symbiosis with B. subtilis on blood agar plates that it was possible to convert it into streptococcus viridans. Other workers had demonstrated that streptococcus hemolyticus possessed a marked affinity for joints; with the change to the viridans type, however, this affinity was lost and the heart valves became the seat of lesions, endocarditis resulting without arthritis. Rosenow showed this differentiation by injecting mixed cultures of these two organisms and mass cultures from tonsils into experimental animals with the result that the organisms were found isolated according to the specific affinity before mentioned, i. e., the viridans group produced endocardial lesions, the hemolytic streptococcus involved the joints.

Rosenow further showed that the same organism might be transformed into a typical, lanceolate, encapsulated, highly virulent pneumococcus producing now, neither endocarditis nor arthritis, but a rapidly fatal pneumococcemia instead. "Further, by the use of shake cultures in test tubes containing a large amount (approximately 12 c.c.) of ascites-dextrose-agar, thus affording a wide range of oxygen pressure, I have been able to isolate the organisms corresponding closely to those described by Poynton and Paine and others, from joint exudate in fourteen out of sixteen cases of acute rheumatic fever. By a similar technic, in which the sediment of blood, after hemolysis had taken place in distilled water, was planted, the organism was isolated from the blood in four out of seven cases, in two cases from stools, and in one from the tonsils. The strain was isolated from the joint in a case of articular rheumatism without muscle involvement. This strain, together with others, formed very long chains and clumps on broth, produced relatively non-adherent large moist colonies surrounded by a zone of green on blood agar plates, and when injected into animals, reproduced the picture of rheumatic fever very closely indeed, producing transient nonsuppurative arthritis, the exudate showing few or no organisms, and a sub-

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endothelial nodular endocarditis in the same animal repeatedly, together with pericarditis in a number of animals. Ulcerative appendicitis, iritis, and conjunctivitis occurred not infrequently after intravenous injection of these strains.

"These strains consist of diplococci and short chains and produce from the beginning a slight hazy and indefinite hemolysis. When these strains, together with other strains of streptococci which are made to resemble these, are injected into animals, they produce an embolic nonsuppurative myositis involving chiefly the flat muscles and more tendinous portion of the muscles of the extremities, a severe myocarditis involving the right ventricle, and arthritis and endocarditis similar to the strains from the cases in which the muscles were not involved. It must not be supposed that the lesions in the muscles are accidental. They occur only at a certain grade of virulence, and at this point the affinity for the muscles is so marked that the number of lesions obtained is in proportion to the size of the dose injected. After one or two animal passages it is again completely lost, and it is now impossible again to produce the muscle lesion.

"A third type resembles those described as micrococcus rheumaticus. They were obtained from cases without muscle involvement, produced small grayish colonies without a green or hemolytic zone, and simple endocarditis and arthritis without myositis and myocarditis when injected intravenously in animals."

Thus we may the more readily comprehend the possibility of malignant endocarditis associated with the presence of streptococcus viridans in blood as a complication of pneumonia or of acute rheumatism since unquestionably the same transmutability is possible within the human body because the same variable degrees of oxygen tension can be found here as in the shake culture employed by Rosenow in his laboratory work. The probable place where this occurs is in the depths of the crypts of the tonsil, etc.

Of course, when the condition complicates puerperal infection which is commonly streptococcic, and other septicemias there is no difficulty in tracing the cause of the endocarditis.

Regarding the mode of entrance, Rosenow, judging largely by the work of Davis and Billings, suggests that the oral cavity most commonly affords the organism admission, and that the order of frequency is here, tonsil, pyorrhea and blind abscesses about the teeth. The lesions in the valves are embolic in origin leading to avascularization of the valves with subsequent breaking down or ulceration of the endocardium, and large cauliflower-like vegetations ensue.

SYMPTOMATOLOGY.

The onset of the disease is so variable in different cases that it has led medicial writers to distinguish between certain types, such as the septic, and the typhoid, and a cardiac and cerebral group. In general one might say, however, that there is an irregular fever, with sweats and a gradual general asthenia with delirium in a large percentage of the cases.

The septic type is usually encountered secondarily to a more or less localized infection elsewhere, such as a tonsillitis, pyorrhea, blind abscess of the teeth, puerperal sepsis, infected wounds, etc. In such cases there is the usual septic type of temperature with big daily variations accompanied frequently by chills and heavy sweats. The heart symptoms are variable and dependent upon the valves involved, or the myocarditis or pericarditis present, and at times may offer little or no evidence of its condition save in the physical findings.

The typhoid type is said to be by far the most common, and is characterized by a fever of more constant type, with lesser remissions, delirium and stupor, profuse sweats and prostration and frequently a goodly number of liquid bowel movements.

Bromwell distinguishes a cardiac group in which patients with chronic valvular lesions give evidence of a recent endocarditis on the old lesion accompanied by acute symptoms, and Osler adds a cerebral group giving the symptoms of a basilar or cerebro-spinal meningitis. Joint symptoms of varying grades of severity are common in all forms. Petechial rashes are common and erythematous ones not infrequent.

The urine may or may not show albumin and casts, but frequently does in the severer types. Streptococci may be found.

The blood findings are variable and while Osler states that there is a marked leukocytosis in infective endocarditis, the cases which I have seen did not show a high grade of leukocytosis at any time, and particularly in the earlier stages, although the differential count showed a relatively high percentage of polymorphoneuclears as the rule. Blood cultures, of course, show a streptococcus in the particular group of cases under consideration.

The course of the disease depends largely upon the type but is almost invariably progressive and while some are of the fulminating rapidly fatal type, others show a remarkable tendency to chronicity as in one case which I have had under observation for some months and in which the onset dates back probably a year from the present time. Again the course may be rapidly changed by the occurrence of embolism, which is quite common and presents variable clinical pictures depending upon the site of lodgment of the embolus. It may involve the central nervous system giving rise to various types of paralysis; infection of the kidney may occur with hematuria. The smaller branches of the mesenteric arteries may become plugged and give rise to ulceration and cause blood to appear in the stools. In one case we saw an acute gastric ulcer with hematemesis, a condition which Rosenow has been able to produce experimentally by the way and from old scars of gastric ulcer he has isolated a streptococcus and diplococcus. In the case of gastric ulcer above recited and concerning which I had some correspondence with Rosenow, he predicted a rapid subsidence of the ulcerative process because of the very low affinity the streptococcus viridans possesses for the mucous mem-

brane of the alimentary tract. His prediction was verified, all evidence of the process disappearing in about 16 or 17 days.

TREATMENT.

The treatment of this condition has been notoriously unsatisfactory, and we must realize that the most to be accomplished is in the way of prophylaxis. The physician must pay more attention to the teeth and tonsils, particularly in cases which give a history of rheumatism or give evidence of old sclerosed valves since these are prone to reinfection.

Regarding the active treatment of the condition, little is to be said of drugs as ordinarily used. Sodium salicylate (and I much prefer the natural wintergreen product to the synthetic salicylate) has been recommended in large doses.

Antistreptococcic serum (polyvalent) is likewise recommended as is the intensive vaccine treatment. An autogenous vaccine is preferable to the stock vaccine and the doses should be increased as rapidly as possible until some effect is obtained, or the dose reaches a thousand million.

Leukocytic extract should be given a thorough trial, and I believe is very helpful.

Another method is the injection intravenously of magnesium sulphate according to the manner of Harrar, who employed it successfully in the treatment of septicemia complicating streptococcus infections of a puerperal nature. This latter process was employed in a case which I saw with Dr. George Reinle and which he will report later. It would seem from our experience in an isolated instance to offer more in the way of treatment of this condition than any of the foregoing remedial measures.

An interesting feature in the use of magnesium sulphate in the case referred to was the change in the blood-picture (an increasing leukocytosis) following each intravenous administration, as will be clearly shown in Dr. Reinle's report of the case.

Through the kindness of Dr. May Sampson, of Berkeley, I am enabled to report the following case:

Miss W. Age 23. Single. Occupation, office nurse for physician. Has been exceptionally well and strong throughout her life and except for measles, escaped the ordinary diseases of childhood. At intervals during the last two or three years has had some distress and pain in right iliac region. Patient thought that for a week or two she had been somewhat sleepy and tired and slightly constipated. No history of typhoid or tuberculosis except for a short association with a relative who had tuberculosis of the throat, and this a period from two or three weeks previous to present illness. Family history is negative.

Present history begins April 7th. While at her usual work she complained of headache with general feeling of lassitude and malaise. Her temperature at this time was 102.8°. An examination of the throat by Dr. F. W. Edmonds in whose employ she was, proved negative. She was sent home and to bed. During the next three days complained still of headache and more or less general soreness and aching. On April 10th she was seen by Dr. May Sampson, of Berkeley. At this time she still complained of the headache, felt chilly and had a slight cough and great thirst. There was no pain. Patient was menstruating nine days before period was due, a thing which had never occurred to her before. Tongue was heavily coated. There was tenderness in the right iliac region without rigidity of abdominal muscles. Examination of chest was negative except for a small patch in the lower lobe posteriorly, which gave a roughened breathing of a bronchial type. Heart was apparently normal. Temperature 105°. Pulse 112. Respirations 32. Blood count 6000 leukocytes; polyneuclears 58%; large mononuclears 8%; small mononuclears 34%.

April 11th. Blood count remained the same. Morning temperature 98°. Pulse 88. Respirations 20. Widal examination by the Berkeley Board of Health was negative; 8 p. m., temperature again rose to 104°.

April 12th. Blood count 6800. Polynuclears 73%. Large mononuclears 8%. Small mononuclears 18%. Urinalysis—Sp. Gr. 1012. Acid. Trace of albumin and many cylindroids. A few pus cells.

At this time Dr. David Hadden was called in because of tenderness persisting in right iliac region. His findings were negative for appendicitis and a diagnosis of a probable typhoid was made.

April 15. Widal made by the Berkeley Board of Health, negative; malaria negative, likewise. Blood count 7200. Large mononuclears 8%. Polynuclears 76%. Small mononuclears 15%.

April 15th. Urinalysis by Western Laboratories showed a trace of albumin and some cylindroids. Diazo absent. Report on the blood from same laboratory was to the effect there was some clumping, but that the loss of motility was not complete, a partial Widal. Blood count 7200. Differential was not made.

April 16th. Berkeley Board of Health reports a partial Widal. Western Laboratories give the same report and negative for para-typhoid. At this time the diagnosis of typhoid seemed fairly well established because of the low blood count, the partial Widals reported by both laboratories, and with more or less tympanites, with gurgling in the right iliac fossa. The spleen, however, was not palpable and there were no rose spots. Heart findings were still negative.

On April 17th a soft systolic murmur was noted in the tricuspid area.

April 20th. Temperature range up to the present time was between 104.4° in the evening and 98° in the morning. On the 11th the morning remission became less, and the evening rise dropped to 102.4° during the next two days following when it again mounted to 104.4° on the evening of the 15th, the morning temperature remaining about 101°. It continued thus for three days when, on the evening of the 18th it reached 105.2°. After this the temperature curve assumed a more characteristic septic type. Widal reported negative by Western Laboratories; partial by Berkeley Board of Health. Blood count in the afternoon 15,400. Polynuclears 88%. At this time it was my privilege to see the case with Dr. Sampson. Patient now complained of photophobia, pain in back of neck, and dyspnea. There was a slight jaundice and the abdomen was tympanitic. The spleen was somewhat enlarged. Examination of chest revealed some dullness in the region of the right lower lobe, a very slight edema of the lungs and a systolic murmur over both tricuspid and aortic areas. Patient had first definite chill during the day, but had had some sweating for a few days previous. Diagnosis of malignant endocarditis was made. Blood culture had been taken in the morning and two c.c. of leukocytic extract had been given. At this time the temperature was 98.2°. Pulse 96. Respirations 28. Cough was more or less troublesome.

April 21st. Blood count at 4:15 p. m. was 9080. Polynuclears 86%. Blood culture revealed a streptococcus. At 1 p. m. there was a chill which lasted half an hour. Cough was quite troublesome and considerable mucus was vomited. Patient was very drowsy and breathing was labored. Tem-

perature had again fluctuated between 104.2° and 102.6°.

April 22nd. Same symptoms persisted. Heart murmurs became louder and rougher and dyspnea more intense. Blood count 8600. Polynuclears 91%.

April 23rd. Blood count 13,400. Polynuclears 82%. Patient's condition about the same. Temperature varied between 101° and 102.8°. Dr. Philip King Brown was called in consultation and diagnosis corroborated. He advised use of polyvalent antistreptococcic serum.

April 24th. The edema of the lungs became more intense, dullness over right lung changed to flatness, manifesting fluid which was later in the day withdrawn.

April 25th. Temperature in morning 99°. Pulse 118. Respirations 54. Cyanosis deepened, dyspnea increased, heart murmur loud. Same symptoms persisted throughout the day and mental confusion became more marked. Pulse became more feeble and there was bronchial breathing, and dullness over left lower lobe. Temperature rose from 99° at noon to 104° at 4 p. m. and remained high. Dyspnea and cyanosis increasing until death occurred shortly after midnight.

Permission was obtained to hold a post mortem which was performed by Dr. Gertrude Moore, whose report is hereto appended:

AUTOPSY ON THE BODY OF MISS W.

An autopsy performed about ten hours after death shows considerable post mortem lividity in the dependent parts with a subcutaneous edema of the lower extremities. The abdomen is distended. Rigor mortis is marked.

The pleural cavities are filled with a blood-stained fluid. The abdominal cavity is well filled with a clear straw-colored fluid. The peritoneal surfaces are everywhere smooth and there are no adhesions. The appendix is normal. The pericardium contains very little more fluid than normal.

In the lower lobes of both the right and left lungs there are irregular, dark red patches which are firm to the touch and do not crepitate. The cut surfaces of these portions of the organ exude a bloody fluid. The remaining portions of the lung are normal.

The heart is slightly larger than normal. The pericardial covering is smooth and glistening with the exception of an area the size of a quarter located on the anterior surface of the left ventricle about one inch above the apex, which is whiter and thicker than the surrounding membrane. The aortic orifice is almost completely closed by a fibrinous mass which is rather firmly attached to two of the valve leaflets. The tricuspid valve is also the site of a fibrinous deposit. On this valve, however, the fibrin is softer than that involving the aortic valve and is less firmly attached. One of the leaflets of the mitral valve is slightly roughened and has a linear deposit of fibrin about 3 mm. from the free edge of the valve.

The liver is somewhat larger than normal, lighter in color and cuts with a decreased resistance. The cut surface is faintly mottled, light areas alternating with darker patches.

The spleen is about one and one-half times its normal size. The capsule is tense and it cuts with decreased resistance. Its pulp is soft and dark red. The kidneys are very slightly increased in size and the capsules strip easily, presenting a surface lighter than normal in color with many small bright red pin-point-sized areas. The cut surface shows a somewhat thickened cortex, the markings of which are lost. The blood vessels are in places considerably engorged, but the parenchymatous tissue of the organ is lighter than normal.

The pelvic organs are normal except for a marked retroversion of the uterus.

ANATOMICAL DIAGNOSIS.

1. Subcutaneous edema.
2. Hydropericardium (slight).
3. Hydrothorax.
4. Hydroperitoneum.
5. Acute lobular pneumonia.
6. Chronic localized pericarditis.
7. Acute valvular endocarditis, aortic, tricuspid, and mitral.
8. Parenchymatous degeneration of the liver (early).
9. Acute splenitis.
10. Acute parenchymatous nephritis (early).

Case II. (Seen with Dr. Geo. Reinle.) Mrs. B. Age 40. Married. Occupation, housewife. Family history negative. Has been exceptionally well and strong throughout her life, except for the diseases of childhood.

Was seen on March 28th, 1914. The history is that on March 26th she was taken suddenly with pains more or less general in her abdomen with some nausea; the following day when seen by me, the pain had localized in McBurney's point, rigidity of muscle was pronounced, blood count leukocytes 27,000, polymorphonuclear 92%, small mononuclear 7%, large mononuclear 1%, eosinophiles 2%, temperature 101.4°. A diagnosis of appendicitis was made and immediate operation advised. Operation performed that same evening; an appendix filled with pus and very adherent was found, which ruptured on being removed. A drain was allowed to remain in the abdomen for one week. Temperature became normal on the third day and remained so until the 10th day of May; patient had been sitting up and about to leave the hospital.

May 11th, patient developed a tonsillitis.

May 12th, 4 a. m., had a slight chill; temperature 103° F. 12 m., chill, vomited, temp. 104.4°. 4 p. m., headache and chill, temp. 101.4°. 12 p. m., chill lasting twenty minutes; temp. 103.6°.

May 13th, 4 a. m., chill lasting ten minutes followed by free perspiration; temp. 100°. 8 a. m., temp. 101°, slight chill. 12 m., chill; temp. 101.2°. 4 p. m., temp. 104°. 8 p. m., temp. 108°. 12 p. m., temp. 103°. Blood count, whites 13,000; polys 75%.

May 14th, 4 a. m., temp. 103.2°, severe chill. 8 a. m., temp. 105°. 12 m., temp. 104.8°. 4 p. m., temp. 105.4°. 8 p. m., temp 103°. 12 p. m., temp. 103°. Blood count, whites 12,000; polys 84%.

May 15th, 4 a. m., temp. 102.8°. 8 a. m., temp. 105.8°. 12 m., temp. 106°. 4 p. m., temp. 104°. 8 p. m., temp. 104.2°. Severe chill followed by patient perspiring freely. 12 p. m., temp. 104°. Blood count, whites 12,000; polys 84%.

May 16th, 4 a. m., temp. 100.4°. 8 a. m., temp. 106°. 12 m., temp. 104°. Chill. 4 p. m., temp. 104°. 8 p. m., temp. 106°. 12 p. m., temp. 104.4°.

May 17th, 4 a. m., temp. 104.8°. Chill. 8 a. m., temp. 104°. 12 m., temp. 103°. 4 p. m., temp. 102.8°. 8 p. m., temp. 104°. At this time 2 c.c. leukocytic extract made by the Western Laboratories of Oakland, was injected intramuscularly and was injected daily until the second day of July.

May 18th, 4 a. m., temp. 103.4°. 8 a. m., temp. 104.6°. Chill. 12 m., temp. 105.6°. 4 p. m., patient perspiring freely; temperature dropped to 101.8°. 8 p. m., temp. 103.4°. 12 p. m., temp. 104°.

May 19th, 8 a. m., temp. 103°. 12 m., temp. 103.4°. At this time a soft systolic murmur was heard in the tricuspid area. 4 p. m., temp. 104.4°. Complains of pain in the left elbow.

May 20th, 8 a. m., temp. 103.4°. Complains of severe pain in right ankle: ankle swollen and tender. 8 p. m., temp. 102°.

May 21st, 8 a. m., temp. 102.6°. 4 p. m., temp. 104°.

May 22nd, 8 a. m., temp. 100.4°. Pain continues severe in right ankle and left elbow. Both joints red, swollen and tender. Blood count, whites 16,000; polys 85%. 4 p. m., temp. 102°.

May 23rd, 8 a. m., temp. 99.8°. 4 p. m., temp. 103.4°.

May 24th, 8 a. m., temp. 101°. 8 p. m., temp. 103.4°.

May 25th, 8 a. m., temp. 101°. Pain continues very severe in leg and arm. 4 p. m., temp. 104.4°.

May 26th, 8 a. m., temp. 102°. 4 p. m., temp. 104.8°. Blood culture taken by Western Laboratories showed many streptococci after forty-eight hours incubation. Blood count, whites 12,000; polys 80%; red count 4,100,000.

May 27th, 8 a. m., temp. 103°. 4 p. m., temp. 104.4°. At this time 400 c.c. 2% C. P. magnesium sulphate solution was injected intravenously; eighteen minutes were consumed in injecting the 400 c.c. solution, injecting 20 c.c. per minute, which seems to me an important part of the technic in this treatment.

May 28th, 8 a. m., temp. 102.8°. 8 p. m., temp. 104.8°. Blood count, whites 16,000; polys 91%.

May 29th, 8 a. m., temp. 102.4°. At noon 400 c.c. 2% magnesium sulphate solution was again injected, following the technic of the previous injection. Blood count, whites 15,400; polys 89%.

May 30th, 8 a. m., temp. 101.6°. 8 p. m., temp. 102.6°. White count 17,200; polys 89%.

May 31st, 8 a. m., temp. 101°. 8 p. m., temp. 102.6°. White count 15,400; polys 82%.

June 1st, 8 a. m., temp. 101.4°. At noon 200 c.c. magnesium sulphate solution was injected. 8 p. m., temp. 101°. White count 16,800; polys 81%.

June 2nd, 8 a. m., temp. 100°. 8 p. m., temp. 103.2°. White count 18,400; polys 84%.

June 3rd, 8 a. m., temp. 99°. 8 p. m., temp. 103°. White count 18,400; polys 84%.

June 4th, 8 a. m., temp. 99°. 8 p. m., temp. 102.4°. White count 20,800; polys 86%.

June 5th, 8 a. m., temp. 99°. 8 p. m., temp. 102°. White count 23,600; polys 87%.

June 6th, 8 a. m., temp. 100°. 8 p. m., temp. 100°. White count 22,400; polys 87%.

June 7th, 8 a. m., temp. 101°. 8 p. m., temp. 103°. White count 21,400; polys 86%.

June 8th, 8 a. m., temp. 99.6°. At noon 400 c.c. magnesium sulphate solution was injected intravenously, followed by nausea. 8 p. m., temp. 104°. White count 23,000; polys 89%.

June 9th, 8 p. m., temp. 101°. 4 p. m., temp. 104.4°.

June 10th, 8 a. m., temp. 100.4°. 8 p. m., temp. 103.6°. White count 22,600; polys 88%.

June 11th, 8 a. m., temp. 99.4°. At noon 400 c.c. magnesium sulphate solution was injected intravenously, followed by a severe chill. Blood culture was taken at this time and remained sterile after seventy-two hours of incubation. 8 p. m., temp. 102.6°.

June 12th, 8 a. m., temp. 99°. 8 p. m., temp. 102°. White count 23,800; polys 92%.

June 13th, 8 a. m., temp. 100°. 8 p. m., temp. 102.4°. White count 23,800; polys 89%.

June 14th, 8 a. m., temp. 99°. 8 p. m., temp. 101°.

June 15th, 8 a. m., temp. 99°. 8 p. m., temp. 101.8°. White count 19,400; polys 84%.

June 16th, 8 a. m., temp. 99.4°. 8 p. m., temp. 101.6°.

June 17th, 8 a. m., temp. 99.4°. 8 p. m., temp. 102.6°. A large abscess had developed in the forearm, which was opened and 500 c.c. of a thin pus was removed.

June 18th, 8 a. m., temp. 99.2°. 8 p. m., temp. 102°.

June 19th, temperature still the same. White count 8,400; polys 84%.

June 20th, temperature the same as preceding day.

June 21st, temperature continues the same. White count 17,800; polys 81%.

June 22nd, white count 18,000; polys 82%. Temperature continues to range between 99° and 102° plus.

June 24th, white count 17,600; polys 91%.

June 26th, white count 17,800; polys 83%.

June 27th, abscess opened right thigh from which 1000 c.c. of pus was removed.

June 29th, temperature 99° and continued so until July 3rd, when temperature reached 103°.

July 4th, 8 a. m., temperature was normal. White count 14,600; polys 87%. On July 9th patient complains of pleurisy of the left side.

July 10th, pain still severe.

July 14th, white count 22,000; polys 89%.

On July 17th left thorax was aspirated, 1000 c.c. clear fluid was removed, when temperature dropped to normal and has remained so ever since.

On July 20th a blood culture was again taken, which remained sterile after seventy-two hours of incubation. The patient has so far entirely recovered, excepting a slight ankylosis of the right knee, due to a septic arthritis. The murmurs in the heart have entirely disappeared.

To recapitulate, this case seems to demonstrate that magnesium sulphate not only destroyed the infection in the blood current, but also increased the leukocytes very materially. Whether this was due to the magnesium sulphate alone, or to the combination of magnesium sulphate and leukocytic extract, we have been unable to tell. According to Harrar of New York, who has used magnesium sulphate in about forty cases of puerperal infection, the action of magnesium sulphate injected intravenously is not known.

This case was a malignant endocarditis, due to a streptococcemia, the infection primarily coming from the tonsils.

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AFTER MEDICINE, WHAT?

By RAYMOND RUSS, M. D.

A friend, a recent graduate who has been filling a long internship in an eastern hospital, has faced a tragedy. He writes that as the result of septicemia he has lost his right hand. His letter is galled by woe and in his helplessness he curses the influences which thrust him into medical work. "My training is purely technical," he has dictated, "to what benefit can I turn knowledge that demands hands as well as head? The years which I have spent in preparation are wasted; yes, worse than wasted, for my heart was in my work and I cannot stifle my interest."

His unhappy state has aroused the pity of all that knew him. Pity is one of the few commodities we bestow with a lavish hand; it is far more difficult to give help. Let us take the measure of the problem. Here is a young fellow after our own hearts. Avidly he has looked forward to medical work, "that full delight," as Stephen Paget puts it, "of pulling people out of death's way." The monotony of the first years had been lightened by the thought of what was to come, and when medical and surgical works placed power within his hands, there came the longing to put his knowledge to the test. The hospital had given added